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The First Plague, and Why It Keeps Coming Back

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16. ABSTRACT

This review paper discusses the history of red tides, their relations to human activities, the chemistry and pharmacology of red tide toxins, the biology of red tide-causing species, and future prospects for red tide research.

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THE RECURRENCE OF THE RED TIDE

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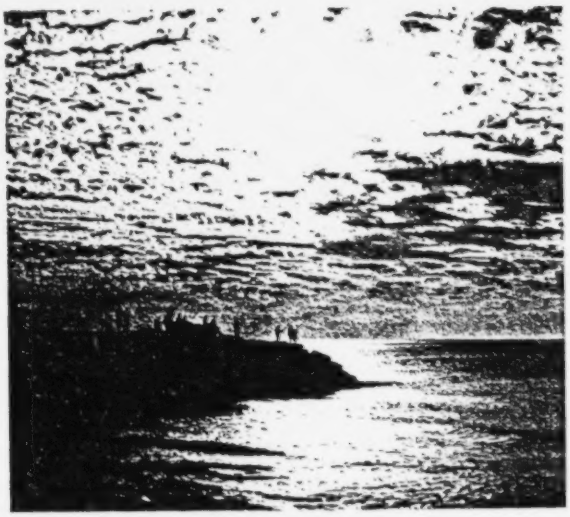

OVERVIEW OF RED TIDE PROBLEMS

As a practical matter, it isn't important whether a catastrophe is natural or manmade, so long as we can predict and prepare for it, avoid it, or prevent it. We think this way about floods, volcanic eruptions, desertification, earthquakes, hurricanes, tornados, urbanization of croplands, and air or water pollution. Our first step always is to understand and explain the phenomenon, and then we proceed to protect ourselves from it. Only after that do we take steps to control or prevent it. We are closer to protection than prevention in our relations with red tides—the first recorded biblical plague upon mankind—those catastrophic overblooms of planktonic algae which kill marine life and even man[4]. These red waters keep coming back, and many believe that we ought to be doing something more about them.[11]

Not all algal blooms are red tides. Not all reddish-amber algal blooms are red tides either. In fact, "red-tide" is not a very definitive term although, like "protoplasm," everyone knows what it describes. In the U.S., the term most often describes blooms of dinoflagellates that harm marine animals or humans. They appear either regularly or sporadically on the East, Gulf, and West coasts of the Continental U.S., in Alaska, Hawaii, and Puerto Rico. Regionally, the causative species differ. Toxicologically, red tide species produce poisons which kill some animals directly and accumulate in the flesh of others, or they may produce no toxin at all. Historically, red tides appear in both pristine and polluted coastal waters. Ecologically, red tide dinoflagellates are a form of marine phytoplankton primary production and are a normal component of the annual plankton cycle of succession. Socially, red tides are a loathsome and mysterious natural phenomenon looked upon with suspicion, fear, and helplessness.

RELATION TO HUMAN ACTIVITIES

As long as there is a question in peoples' minds about a relation of red tides to pollution, then red tide remains a pollution issue. Dinoflagellate blooms affect mankind significantly in economic, social, scientific, medical, legal, and even artistic ways. Many dinoflagellate species luminesce[7,8], and their flashes of light in the night seas have inspired poetry and paintings. Economic impacts of red tides may be devastating, and relate to our need to protect ourselves from toxic dinoflagellate blooms by quarantine of the infested waters—closing them to shellfish harvest, finfishing, and water contact sports. Such quarantine produces social effects directly through removal of recreational outlets and indirectly through economic effects on fishing and recreational industries. Perhaps we wrongly associate red tides with pollution because most shellfish bed closures presently

are based on criteria of bacterial contamination caused by sewage overflows. Red tides enter the law legitimately through the need for government to use its regulatory and enforcement police powers to protect the public from health hazards of paralytic nerve poison produced by many dinoflagellates. Medicine treats stricken victims if there is time for diagnosis, and science delves into causative factors of red tides, and ways to predict and eventually control them—perhaps even turning them to beneficial purposes.

At EPA adjudicatory hearings concerning a discharge permit for the Seabrook Nuclear Power Plant under construction in New Hampshire, a witness opposed to the permit raised an unique question in regard to industrial cooling water use [14]. His concern was that the cooling water, warmed and discharged into pollutant enriched waters derived from the Merrimac River a few miles to the north, would create conditions favorable for the formation of red tides. Given that red tides recently had appeared in New England waters and that they are fostered by warmth, shallowness, and nutrient enrichments, it seemed a reasonable thought, and EPA considered it carefully. This led to a consideration of the balance between conditions which favored a red tide bloom and conditions which acted to prevent one.

Studies of what regulated the onset of winter-spring phytoplankton blooms in Narragansett Bay over four and one-half years showed that their onset there was not due to water mass stability, light, or temperature, but rather to release of grazing pressure by zooplankton—which itself had been determined by the proceeding crop of phytoplankton. Other studies in an artificial stream showed that increases in temperature alone caused only insignificant increases in phytoplankton productivity whereas significant increases were due to other physical and chemical factors tested. There is insufficient knowledge of dinoflagellate environmental requirement differences on a species by species basis to predict a given species' dominance even under known changes of physical, chemical, and nutritional factors. Further, successions within an ecological system are complicated by the presence and activity in marine ecosystems of naturally occurring antimetabolites which prevent proliferation of the red tide species[6].

Seabrook's industrial cooling waters could be expected to encourage proliferation of red tide dinoflagellates only if several conditions were met. The abundant presence of reproductive stages of the dinoflagellate species would have to coincide with the following; an absence of naturally occurring antimetabolites, the availability of adequate and proper nutrients, an actual temperature increase favoring the red tide species and persisting long enough to maintain an advantage for it, and the absence of heavy grazing pressure from zooplankton in the area. EPA reasoned as follows[14]: "Due to the distance of large volumes of nutrient input from the source of heated discharge and the transient nature of that discharge's thermal component, the possibility of red tide conditions being created by the interaction of heat and nutrients in this instance seems very remote, but not impossible. There is, however, no available evidence of red tide proliferation in association with any electrical generating facility in New England, even among those plants that operate routinely in over-enriched waters."

The catastrophic New York Bight dinoflagellate bloom of 1976 was an entirely different matter in which natural and manmade forces combined to kill marine life over an estimated 70 mile stretch of coastline[9]. Its greatest areal extent covered roughly half the New York Bight, including all waters east of New Jersey and northern Delaware and, by September of 1976, extended some 40 miles offshore. Although the dominant species, *Ceratium tripos*, does not produce a toxin, its presence in such numbers created a biological oxygen demand so great that the pockets of anoxic conditions which it caused killed crabs, fishes, clams, lobsters, and all marine life that could not escape. Mid-September estimates were that 59,000 metric tons of surf clam meats (not including shells) or 25% of the New Jersey offshore clam stocks were lost.

The late spring through autumn of 1976 saw unusual meteorological and hydrological conditions in the New York Bight. Winds and currents acted to hold unusually high polluted river runoffs in the area, and water temperatures were unusually high for the season. Offshore waters containing *C. tripos* intruded into the area displacing shoreward bottom waters. Of some 45 million people inhabiting the mid-Atlantic Bight (southern New England to northern Virginia), about half dispose of wastes into the waters of the New York Bight. All of the necessary and sufficient conditions for red tide formations came together, and the Atlantic's largest recorded red tide occurred. Had the bloom species been toxic *Gonyaulax excavata*, which has poisoned shellfish in New England and also inhabits the New York Bight waters, many persons might have been among the red tide victims. Red tide should be expected to return to the New York Bight on some future sustained southwesterly wind after a warm, storm-free springtime. All of the other factors are there, waiting in the waters to plague us again[2].

RED TIDE TOXINS

Toxic red tides may have effects on humans far removed from ocean waters because their toxins are accumulated by edible marine shellfish. The disease they cause is known as paralytic shellfish poisoning (PSP). Shellfish acquire toxicity by ingesting dinoflagellate cells containing endotoxins, and incorporating the endotoxins into their own flesh[10]. Certain finfish accumulate dinoflagellate toxins similarly, by grazing on algae covered with dinoflagellate cysts, and when eaten cause the same symptomology; numbness within minutes, respiratory irritation and distress, labored breathing, paralysis, and death from anoxia within 24 hours—or recovery with no after effects (except, perhaps, a much greater willingness to remember whether one does or

does not eat certain seafoods taken during months spelled with an "r")[3].

The chemical structure of PSP, sought since the first rough chemical isolations from the Alaska butter clam and from axenic (single species, bacteria-free) cultures of *Gonyaulax catenella* in the 1950's, now is known for two of the three most common red tide dinoflagellates[5,10,11]. The molecular formula of the isolated hydrochloride salt is $C_{10}H_{17}N_7O_4$. 2HCl and its molecular weight is 372. It is a 3, 4, 6 trialkyltetrahydropurine and its structure is confirmed by synthesis and subsequent biological tests which confirm its activity. The poison is a strong base having one pK_a at 11.5 and a second at 8.2. It has no ultraviolet absorption, gives positive Benedict-Behre and Jaffe tests, and is detoxified completely by mild catalytic reduction. This poison occurs also in cells of *Gonyaulax tamarensis* (the north Atlantic toxic red tide species, also recently identified as *Gonyaulax excavata*), which produces several additional toxic entities that chemically are not fully identified.

Gymnodinium breve, a south Atlantic and Gulf Coast red tide dinoflagellate, also produces separable toxic entites[7,10,11]. These are relatively polar lipid compounds for which infrared, visible, and ultraviolet spectroscopic analysis suggest aromatic and carbonyl radicals. Infrared and mass spectroscopy show that the toxins have some relationships to dialkyl phthalate compounds, although this probably is due artifactually to plastics derived contaminants from the toxin isolation procedures. Much remains unknown about *G. breve* toxins[11].

All of the chemical isolations of various dinoflagellate (and other algal) toxins involve some form of chromatographic separation under acidic conditions[7,10,11]. Unfortunately, no two teams of investigators chromatographed their materials the same way, or chose the same mobile phase solvent mixtures. One and two dimensional thin layer chromatography, column chromatography (all using different solvents), and glass capillary chromatography, produce curiously different chemical isolates of variable purity and questionable relationship to the naturally occurring molecule. No matter how standard the detection devices, different chromatography procedures must yield different and therefore confusing analyses of the toxins. Further confounding the understanding of toxin structure are variable biological source materials, extraction procedures, and biological effect tests. A synthesis of existing knowledge and some standardization of methods must occur before apparent chemical differences among dinoflagellate toxins are understood.

Pharmacologically, dinoflagellate toxins burst mammalian blood cells, prevent clotting, and interfere with nerve impulse generation and passage in man, fishes, frogs, mice, and molluscan shellfishes[11]. There is a direct relationship between a marine animal's own resistance to dinoflagellate toxin and the danger to man of eating a resistant (and therefore highly toxin-impregnated) animal. Thus the east coast oyster, *Crassostrea virginica*, is very sensitive to its indigenous dinoflagellate's toxin, and never has been implicated in a human case of PSP. Conversely, the soft shell clam, *Mya arenaria*, the blue mussel, *Mytilus edulis*, and the sea scallop, *Placopectin magellanicus*, all are quite resistant to their indigenous red tide toxins, and all have caused human PSP fatalities[4]. Resistant animals exposed to red tides have accumulated from 1000 to 10,000 μg of toxin in 100 g of flesh.

What we know about marine animals' accumulation of dinoflagellate endotoxins is derived from human experience and a very few scientific studies[1,13]. Some reasons for animals' different susceptibilities to red tide toxins and different abilities to accumulate it in their flesh are now under study[4]. These are: genetic differences in nerve cell vulnerability to the toxins, differences in physiological activity among the toxins themselves, behavioral differences in feeding among exposed animals, habitat differences causing differential animal exposure, and differences in animals' ability to store toxins in physiologically isolated parts of their bodies. For human safety precaution, the quarantine concentration of PSP toxin for edible flesh of marine life is set low, at 80 μg/100 g—presumably to account for individual differences in vulnerability among people.

The mechanism of red tide neurotoxin activity is much more thoroughly studied, at least for the chemically known *Gonyaulax excavata* and *Gonyaulax catenella* toxins[4,10]. Nerve impulse transmission is an electrochemical phenomenon which occurs across nerve cell membranes. At rest, the nerve axon membrane maintains an electrical potential due to outside higher sodium ion concentration and inside higher potassium ion concentration. The inside is negative relative to the positive outside of the nerve. To transmit an active electrical nerve impulse, a stimulus in the form of an outward electrical impulse must occur. When it does, the membrane becomes permeable to sodium ions progressively and regeneratively along its length. This creates a measurable electrical impulse. As the impulse travels along the nerve, potassium permeability increases behind it, restoring the nerve's resting potential so that it may transmit the next electrical impulse by chemical means. *G. tamarensis* toxins act by blocking the transmission of electrochemical impulses in the nerve. A cationic form of toxin binds reversably with the external surface of the membrane, stoichiometrically blocking the membrane sites of sodium permeability. Without its sodium permeability, the nerve cannot conduct an electrical impulse. It is this failure of nerve impulse conduction that accounts for the paralytic effects of dinoflagellate toxins.

In addition to its neurotoxic effects, *Gymnodinium breve* toxin contains fractions with hemolytic activity, anticoagulant activity, mild contact dermatitis activity, and which act as a severe respiratory irritant[4,11]. The hemolytic activity is slight for mammalian blood, but autopsied fishes killed in a Florida red tide showed acute tissue damage, hemolysis, and interference with blood clotting mechanisms. *G. Breve* toxins are the least understood of all dinofla-

gellate toxins, chemically, pharmacologically, and biologically. This is due in part to the difficulty of growing *G. breve* in laboratory culture (as compared to the ease of growing other toxic algal species), in part to the difficulty of studying red tides opportunistically in the field, and in part to real chemical differences in its toxins from those of other algae.

BIOLOGY OF RED TIDE SPECIES

Early warning of catastrophic red tides through seawater and shellfish monitoring and protection from them by legal and medical means now are within our technical abilities. Prevention of red tides is not, probably because we don't know enough about the environmental factors required to be present and required to be absent as necessary and sufficient for a red tide species to dominate and persist in the plankton community. The literature on dinoflagellate physiology and nutrition amounts to hundreds of articles and several reviews.

Dinoflagellates are highly complex single celled creatures with both truly plant and truly animal species. The most common red tide species possess characteristics of both plants and animals—photosynthesis and nutritional requirements for certain organic molecules[6]. Almost all dinoflagellates studied require vitamins B12, biotin, and thiamine as coenzymes for growth and reproduction. Many photosynthetic species can use organic sources of nitrogen and carbon, although they grow nearly as well in sterile synthetic media containing only vitamins, salts and trace elements of seawater, and inorganic nitrogen, carbon, sulfur and phosphorous sources. Their nitrogen metabolism may differ from that of most plants in that the presence of more reduced forms of nitrogen does not suppress enzyme activity needed to assimilate less reduced nitrogen sources[4]. This broader ability to use diverse nitrogen sources should be an adaptive advantage offshore, where nitrogen often is the limiting factor for surface phytoplankton growth.

Environments plagued by red tides differ. In the northeast Atlantic's Bay of Fundy and in the Pacific northwest, waters are cold and deep. Their main nutrient sources come from upwellings of deep ocean water near shore. In the south Atlantic and Gulf, waters are warm and shallow, with land runoff providing nutrients. The mid Atlantic Bight derives nutrients for its broad continental shelf waters from both land and oceanic sources. Red tide dinoflagellates in northern waters are offshore species requiring relatively high salinities to grow and reproduce[19]. This appears to be true of *G. breve* in Florida also. However, there is recent evidence that red tide blooms originate in inshore estuaries and proceed outward as viable cysts when estuarine sediments are resuspended into the water column[2]. Dinoflagellates can migrate vertically in the water column over tens of meters—downward at night and upward during the daylight. This is an adaptive advantage for them over diatoms and other algae with which they must compete for soluble nutrients. All can photosynthesize during the day, but at night dinoflagellates can use reduced nitrogen sources found at greater depths. This may compensate in part for their relatively slow reproductive doubling time, and accounts for red tide blooms in surface waters which are nutrient poor. Vertical migration serves also as a means of recruitment by which offshore species of dinoflagellates may enter inshore waters, migrating upward out of a tidal salt wedge intrusion under nearshore waters during daylight hours.

Dinoflagellates are not very sensitive to temperature differences, and are limited by temperature only in geographic distribution of species[6]. Within broad temperature tolerances, warming of the water causes more rapid cell divisions and thereby fosters bloom conditions favorable to red tide species. Under unfavorable conditions, dinoflagellates may secrete a cyst wall around themselves and become dormant. They survive in encysted form over winter or during periods of nutrient depletion (seen in neglected laboratory cultures). Factors which cause encystment and excystment are not well understood. Extreme cold temperature produces heavy walled overwintering cysts but nutrient depletion produces thin walled, short term cysts in *G. tamarensis*[2]. In the free swimming stage of life, dinoflagellates reproduce by longitudinal fission. Encysted, dinoflagellates may reproduce by longitudinal or by multiple fission. There is an option of sexual reproduction as well for some dinoflagellate species, but dinoflagellate life cycles in nature differ considerably among species[14,17]. None of the red tide species' life cycles are completely understood, and there remains much surmise about the natural origin of red tides.

Microscopic observations of *G. breve* exposed to low concentrations of ionic heavy metals show that cells round-up, throw off their flagella, and swell until each cell bursts. From early 1950's through mid 1960's the U.S. Bureau of Commercial Fisheries tested $CuSO_4$ and numerous other materials at sea for their ability to kill *G. breve* selectively[13]. The conclusion of extensive field testing was that $CuSO_4$ could provide temporary red tide prevention in localized areas, but its effects were too transient to be considered feasible for large scale relief from red tides. Thus far the literature records just this one attempt to prevent or abate red tide formation in nature. However, anyone who knows the fragility of *G. breve* and most other dinoflagellate cells in culture has little trouble believing that some feasible means of field control simply await recognition[4,12].

FUTURE RED TIDES AND RED TIDE RESEARCH

Vladimir Nabakov wrote, "Without fantasy there is no science, and without fact there is no art." This is a great comfort when one attempts to extrapolate from solid, peer reviewed science into the future, hoping not to trespass upon science fiction in the process. Perhaps a solution to the red tide prevention problem will come quicker from heuristic than from deduction approaches. Just as agriculture has

turned to chemistry for a spectrum of weed killers, so mariculture may one day find some compound which acts selectively against red tide dinoflagellates. Meanwhile, no one seems to be looking for anti-red tide materials as such.

Are we fostering the return of red tide by pouring hundreds of millions of gallons of treated and partially treated domestic and industrial sewage into our coastal waters? Oddly enough, only one study done in Norway nearly 30 years ago addressed this question directly. It found that low concentrations of sewage stimulated dinoflagellate growth but high concentrations were inhibitory[3]. Although very limited, this early observation agrees with current knowledge of sewage composition and dinoflagellate physiology—heavy metals and high organic content limiting dinoflagellate populations, and small amounts of organic nutrients proving beneficial to their growth and reproduction. The New York Bight red tide of 1976 indicates but does not prove that confined, dilute pollutants create a favorable situation for red tide formation. Absence of red tides' regular recurrence in the annual phytoplankton successions indicates the extent to which red tides are dependent upon naturally varying biological and hydrological factors[4]. We certainly are providing enough organic nutrients in usable form, as shown by blooms of other nuisance algal species, in most of our urban bays and estuaries. Are these other species more resistant to heavy metals and synthetic organic wastes in sewage, or do they utilize sewage nutrients more efficiently than red tide dinoflagellates? Probably both[4]. Do red tide species require seawater that is depleted of even its normal concentrations of trace heavy metals? This seems likely, as red tides appear in association with natural and contaminant organic chelating materials, and reducing conditions[4,6,9]. Our attempts to recover and recycle domestic and industrial wastes should eventually remove red tides from the lists of possible pollutant effects—at least to the extent to which they are successful. Meanwhile, we should continue laboratory studies of dinoflagellate environmental requirements, life cycles, toxin production, and vulnerabilities to test new or modified existing hypotheses on the origin and prevention of red tides.

Red tide toxins themselves may have uses to explore. The chemically identified trialkyl-tetrahydropurine toxin which blocks nerve membrane sites of sodium permeability does so 1:1 stoichiometrically. This provides a tool to study the relative density of various nerves' sodium permeability sites which may be useful in comparative neurophysiology. As a minimum the toxin is useful to identify and characterize the sodium permeability of nerve membrane, and as a site specific sodium block for other experimental purposes. Similar uses for the chemically elusive *G. breve* toxins are likely if they present any new structural activities upon purification, identification, and pharmacological examination of their several poisonous effects.

One other red tide research future on the horizon is a by product of the Department of Energy's "Ocean Thermal Energy Conversion" (OTEC) program. The OTEC concept involves using the temperature difference between warm surface waters and cold bottom waters as a source of energy to generate electric power. Small pilot plant studies are underway now and commercial OTEC generation is projected for the 1990's. The OTEC cooling water flow required is 10X that of presently designed steam thermoelectric generating plants. Ten thousand gpm/megawatt represents a large-scale upwelling of nutrient rich bottom waters—a condition often associated with red tide formation. Effects of OTEC water movement on deepwater and surface dwelling organisms is an identified study need in the current 5 year "Federal Plan for Ocean Pollution Research, Development, and Monitoring[18]."

REFERENCES:

1. Alam, M.E., C.P. Hsu, and Y. Shimizu, 1979. Comparison of Toxins in Three Isolates of Gonyaulax tamarensis (Dinophyceae). Jour. Phycology 15(1):106-110.

2. Anderson, D.M., and D. Wall, 1978. Potential Importance of Benthic Cysts of Gonyaulax tamarensis and G. excavata in Initiating Toxic Dinoflagellate Blooms. Jour. Phycology 14(2):224-234.

3. Braarud, T., and Pappas, I., 1951. Experimental Studies on the Dinoflagellate Peridinium triquetrum Ehrb. (Lebour). Norske Videnshaps-Akad. Oslo I. Mat-Naturv. Klasse No. 2:1-23.

4. Holy Scriptures: Exodus: 7:20-21

5. LoCicero, Vincent R. (Ed.)., 1975. Proceedings of the First International Conference on Toxic Dinoflagellate Blooms. Mass. Science and Technology Foundation, Wakefield, MA.

6. Prager, Jan C., 1974. Factors Influencing Estuarine Microbiota. Chapter 4 in Sax, N.I. (Ed.). "Industrial Pollution", Van Nostrand Reinhold Co., N.Y.

7. Scheuer, P.J., 1973. Chemistry of Marine Natural Products. Academic Press, N.Y.

8. Schmidt, R.J., V.D. Gooch, A.R. Loeblich III., and J.W. Hastings, 1978. Comparative Study of Luminescent and Nonluminescent Strains of Gonyaulax excavata (Pyrrhophyta). Jour. Phycology, 14(1):5-9.

9. Sharp, Jonathan H. (Ed.)., 1976. Anoxia on the Middle Atlantic Shelf During the Summer of 1976. Workshop Report. National Science Foundation, IDOE, Wash., D.C.

10. Shimizu, Y., 1979. Dinoflagellate Toxins. Chapter 1 in P.J. Scheuer, (Ed.), Marine Natural Products, Chemical and Biological Perspectives. Academic Press, N.Y.

11. Steidinger, K., Burklew, M.A., and R.M. Ingle, 1973. The Effects of Gymnodinium breve Toxin on Estuarine Animals. Chapter 6 in D.F. Martin and G.M. Padilla (Eds.), Marine Pharmacognosy, Action of Marine Biotoxins at the Cellular Level. Academic Press, N.Y.

12. Steidinger, K.A., E.W. Truby, and C.J. Dawes, 1978. Ultrastructure of the Red Tide Dinoflagellate. I. General Description. Jour. Phycology 14(1):72-79.

13. Targett, N.M., and A. Mitsui, 1979. Toxicity of Subtropical Marine Algae Using Fish Mortality and Red Blood Cell Hemolysis for Bioassays. Jour. Phycology 15(2):181-185.

14. Turpin, D.H., P.E.R. Dobell, and F.J.R. Taylor, 1978. Sexuality and Cyst Formation in Pacific Strains of the Toxic Dinoflagellate Gonyaulax tamarensis. Jour. Phycology 14(2):235-238.

15. U.S. Dept. of Interior, 1965. Bureau of Commercial Fisheries Symposium on Red Tide. SSR. No. 521.

16. U.S. Environmental Protection Agency, 1976. Proceedings of the Adjudicatory Hearing on NPDES Permit Application No. NH0020338. Exhibit 39. pp. 1-18. U.S.E.P.A. Region I. Boston, MA.

17. Walker, L.M., and K.A. Steidinger, 1979. Sexual Reproduction in the Dinoflagellate Gonyaulax monilata. Jour. Phycology 15(3):312-315.

18. Walsh, James P. and Gage, Stephen., 1979. Federal Plan for Ocean Pollution Research, Development, and Monitoring. Fiscal Years 1979-1983. NOAA. National Technical Information Service, Springfield, VA.

19. White, A.W., 1978. Salinity Effects on Growth and Toxin Content of Gonyaulax excavata, a Marine Dinoflagellate Causing Paralytic Shellfish Poisoning. Jour. Phycology 14(4):475-479.

turned to chemistry for a spectrum of weed killers, so mariculture may one day find some compound which acts selectively against red tide dinoflagellates. Meanwhile, no one seems to be looking for anti-red tide materials as such.

Are we fostering the return of red tide by pouring hundreds of millions of gallons of treated and partially treated domestic and industrial sewage into our coastal waters? Oddly enough, only one study done in Norway nearly 30 years ago addressed this question directly. It found that low concentrations of sewage stimulated dinoflagellate growth but high concentrations were inhibitory[3]. Although very limited, this early observation agrees with current knowledge of sewage composition and dinoflagellate physiology—heavy metals and high organic content limiting dinoflagellate populations, and small amounts of organic nutrients proving beneficial to their growth and reproduction. The New York Bight red tide of 1976 indicates but does not prove that confined, dilute pollutants create a favorable situation for red tide formation. Absence of red tides' regular recurrence in the annual phytoplankton successions indicates the extent to which red tides are dependent upon naturally varying biological and hydrological factors[6]. We certainly are providing enough organic nutrients in usable form, as shown by blooms of other nuisance algal species, in most of our urban bays and estuaries. Are these other species more resistant to heavy metals and synthetic organic wastes in sewage, or do they utilize sewage nutrients more efficiently than red tide dinoflagellates? Probably both[6]. Do red tide species require seawater that is depleted of even its normal concentrations of trace heavy metals? This seems likely, as red tides appear in association with natural and contaminant organic chelating materials, and reducing conditions[4,6,9]. Our attempts to recover and recycle domestic and industrial wastes should eventually remove red tides from the lists of possible pollutant effects—at least to the extent to which they are successful. Meanwhile, we should continue laboratory studies of dinoflagellate environmental requirements, life cycles, toxin production, and vulnerabilities to test new or modified existing hypotheses on the origin and prevention of red tides.

Red tide toxins themselves may have uses to explore. The chemically identified trialkyl-tetrahydropurine toxin which blocks nerve membrane sites of sodium permeability does so 1:1 stoichiometrically. This provides a tool to study the relative density of various nerves' sodium permeability sites which may be useful in comparative neurophysiology. As a minimum the toxin is useful to identify and characterize the sodium permeability of nerve membrane, and as a site specific sodium block for other experimental purposes. Similar uses for the chemically elusive *G. breve* toxins are likely if they present any new structural activities upon purification, identification, and pharmacological examination of their several poisonous effects.

One other red tide research future on the horizon is a by product of the Department of Energy's "Ocean Thermal Energy Conversion" (OTEC) program. The OTEC concept involves using the temperature difference between warm surface waters and cold bottom waters as a source of energy to generate electric power. Small pilot plant studies are underway now and commercial OTEC generation is projected for the 1990's. The OTEC cooling water flow required is 10X that of presently designed steam thermoelectric generating plants. Ten thousand gpm/megawatt represents a large-scale upwelling of nutrient rich bottom waters—a condition often associated with red tide formation. Effects of OTEC water movement on deepwater and surface dwelling organisms is an identified study need in the current 5 year "Federal Plan for Ocean Pollution Research, Development, and Monitoring[18]."

REFERENCES:

1. Alam, M.E., C.P. Hsu, and Y. Shimizu, 1979. Comparison of Toxins in Three Isolates of Gonyaulax tamarensis (Dinophyceae). Jour. Phycology 15(1):106-110.

2. Anderson, D.M., and D. Wall, 1978. Potential Importance of Benthic Cysts of Gonyaulax tamarensis and G. excavata in Initiating Toxic Dinoflagellate Blooms. Jour. Phycology 14(2):224-234.

3. Braarud, T., and Pappas, I., 1951. Experimental Studies on the Dinoflagellate Peridinium triquetrum Ehrb. (Lebour). Norske Videnshaps-Akad. Oslo I. Mat-Naturv. Klasse No. 2:1-23.

4. Holy Scriptures: Exodus: 7:20-21

5. LoCicero, Vincent R. (Ed.)., 1975. Proceedings of the First International Conference on Toxic Dinoflagellate Blooms. Mass. Science and Technology Foundation, Wakefield, MA.

6. Prager, Jan C., 1974. Factors Influencing Estuarine Microbiota. Chapter 4 in Sax, N.I. (Ed.). "Industrial Pollution", Van Nostrand Reinhold Co., N.Y.

7. Scheuer, P.J., 1973. Chemistry of Marine Natural Products. Academic Press, N.Y.

8. Schmidt, R.J., V.D. Gooch, A.R. Loeblich III., and J.W. Hastings, 1978. Comparative Study of Luminescent and Nonluminescent Strains of Gonyaulax excavata (Pyrrhophyta). Jour. Phycology, 14(1):5-9.

9. Sharp, Jonathan H. (Ed.)., 1976. Anoxia on the Middle Atlantic Shelf During the Summer of 1976. Workshop Report. National Science Foundation, IDOE, Wash., D.C.

10. Shimizu, Y., 1979. Dinoflagellate Toxins. Chapter 1 in P.J. Scheuer, (Ed.), Marine Natural Products, Chemical and Biological Perspectives. Academic Press, N.Y.

11. Steidinger, K., Burklew, M.A., and R.M. Ingle, 1973. The Effects of Gymnodinium breve Toxin on Estuarine Animals. Chapter 6 in D.F. Martin and G.M. Padilla (Eds.), Marine Pharmacognosy, Action of Marine Biotoxins at the Cellular Level. Academic Press, N.Y.

12. Steidinger, K.A., E.W. Truby, and C.J. Dawes, 1978. Ultrastructure of the Red Tide Dinoflagellate. I. General Description. Jour. Phycology 14(1):72-79.

13. Targett, N.M., and A. Mitsui, 1979. Toxicity of Subtropical Marine Algae Using Fish Mortality and Red Blood Cell Hemolysis for Bioassays. Jour. Phycology 15(2):181-185.

14. Turpin, D.H., P.E.R. Dobell, and F.J.R. Taylor, 1978. Sexuality and Cyst Formation in Pacific Strains of the Toxic Dinoflagellate Gonyaulax tamarensis. Jour. Phycology 14(2):235-238.

15. U.S. Dept. of Interior, 1965. Bureau of Commercial Fisheries Symposium on Red Tide. SSR. No. 521.

16. U.S. Environmental Protection Agency, 1976. Proceedings of the Adjudicatory Hearing on NPDES Permit Application No. NH0020338. Exhibit 39. pp. 1-18. U.S.E.P.A. Region I. Boston, MA.

17. Walker, L.M., and K.A. Steidinger, 1979. Sexual Reproduction in the Dinoflagellate Gonyaulax monilata. Jour. Phycology 15(3):312-315.

18. Walsh, James P. and Gage, Stephen., 1979. Federal Plan for Ocean Pollution Research, Development, and Monitoring. Fiscal Years 1979-1983. NOAA. National Technical Information Service, Springfield, VA.

19. White, A.W., 1978. Salinity Effects on Growth and Toxin Content of Gonyaulax excavata, a Marine Dinoflagellate Causing Paralytic Shellfish Poisoning. Jour. Phycology 14(4):475-479.